1970). The following results were observed:

- 1) The frequency of lethal genes in the natural population was 32.5% for the third chromosome and 8.3% for the second.
- 2) In the cage population the frequencies of both second and third chromosome lethals were at the same level, 11.7%.
- 3) No significant differences were observed in sl  $\pm$  sv genes on both chromosomes in the cage and the natural populations.
- 4) Between 14 Cy/1 genotypes in the cage population, 45 (observed) crosses were made and 6 (with the frequency of 13.3%) were observed to be alleles. The same percentage was calculated for the third chromosome. From the natural population, 2 alleles for the second and 18 for the third chromosomes were observed. The frequencies were 4.4% and 7.2% respectively. All flies were raised in Mostashfi medium and reared at  $23 \pm 1^{\circ}\text{C}$ .

Conclusion: In natural populations of D. melanogaster in Tehran, the number of lethal genes on the third chromosome is higher than the second chromosome. In cage populations (set 15 years ago), the frequencies of lethal genes on both major autosomes are in equilibrium. No seasonal changes are observed in the natural population of Tehran.

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effect variegation of Pgd locus determining 6-phosphogluconate dehydrogenase
in Drosophila melanogaster.

The structural gene Pgd for 6-phosphogluconate dehydrogenase (6PGD) was located at 0.65 to the left of pn locus of the X-chromosome of D. melanogaster (1). Position effect variegation of Pgd locus has been studied in the duplication Dp(1;f)R carrying the PgdA allele. This duplication covers y to kz (according to Schultz's data). Flies were cultivated at  $18^{\circ}$  in order

to increase the extent of variegation. We have obtained the marked variegation for sc, dor and pn loci for XO males only. The specific activity of 6PGD in these mosaic XO males was compared with that of non-mosaic XY males. The results demonstrate that the variegation for dor, sc and pn correlates with the decrease of 6PGD activity. Various genotypes and X-chromosomes were used for the study of the position effect for Pgd locus (see Table). In the first two series of experiments the specific activity of 6PGD of XO males has been diminished approximately to 80% level of XY males. In the third series with X-chromosome lacking the Pgd locus (Pgd-kz deficiency) the enzyme activity of XO mosaics was reduced to 70% level of non-mosaic XY males. In this latter series the genotype of mosaic males differed from that of non-mosaics by the absence of Y-chromosome only.

The ratio of 6PGD specific activities of mosaic (XO) to non-mosaic (XY) males.

Series of exp.	No of tests	Markers of X-chromosome	Character of variegation	XO;XY ratio of 6PGD activities
1	11	y dor <sup>l</sup> Pgd <sup>A</sup>	$dor^{V}$	0.79 ± 0.03
2	13	y ac sc Pgd $^{ m B}$ w	$sc^{\mathbf{v}}$	$0.83 \pm 0.02$
3	15	Df(1)Pgd-kz (Pgd-pn-)	$pn^V$	$0.71 \pm 0.05$

The elimination of Y chromosome from normal males without rearrangements leads to 20% increase of 6PGD activity. Therefore the absence of Y chromosome masked the true decrease of Pgd locus inactivation induced by position effect. The isozyme patterns of 6PGD obtained in acryl amide gel electrophoresis of crude extracts of XO and XY males carrying  $Pgd^B$  in the X-chromosome and  $Pgd^A$  in duplication Dp(1;f)R have been compared (see Table, the second series of experiments). The  $Pgd^A$  allele determines the fast isozyme of 6PGD and the  $Pgd^B$  the slow one (2). In the XO males the activities of the fast and hybrid isozymes were diminished while that of the slow one was not affected. We interpret the decrease of the total 6PGD activity and the change of isozyme pattern in phenotypically expressed mosaics as result of variegated position effect of Pgd locus.

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